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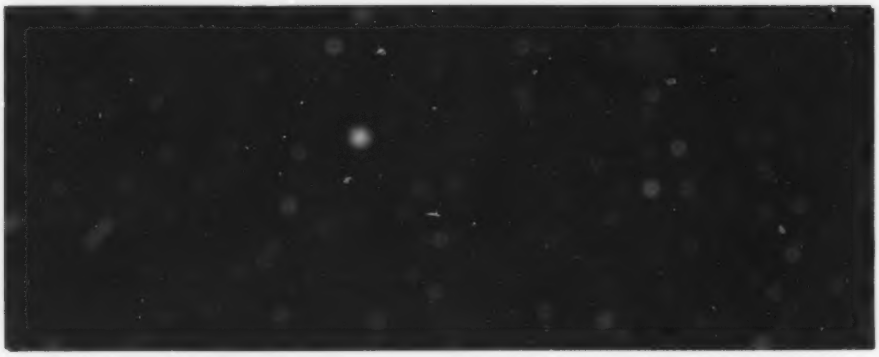

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Final Report

R-86-05-0171

Studies on Resistance in Wheat to Septoria Nodorum Blotch and Tan Spot

Conducted by
G.R. Hughes

Funded by the Agriculture Development Fund

R-86-05-0171

STUDIES ON RESISTANCE IN WHEAT TO SEPTORIA NODORUM BLOTCH AND TAN SPOT

Project R-86-05-0171

FINAL REPORT

submitted to

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Saskatchewan Agriculture

by

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ABSTRACT

Sources of resistance to septoria nodorum blotch, caused by Leptosphaeria nodorum, and to tan spot, caused by Pyrenophora tritici-repentis, were identified. Resistance to L. nodorum in 12 cultivars was under single gene control and allelism studies indicated at least two different genes exist. Seedling and field disease reaction was highly correlated and broad-sense heritability estimates ranged from 71 to 90%. No clear evidence for physiologic specialization among Saskatchewan isolates of L. nodorum was found, but isolates differed in pathogenicity. Resistance to P. tritici-repentis in three cultivars was also monogenically controlled. Cultivar 81IWMMN 2095 possessed excellent resistance to both pathogens. Development of varieties resistant to septoria nodorum blotch, an important disease of wheat in Saskatchewan, is in progress.

SUMMARY

This study was undertaken to provide information necessary for breeding for resistance to septoria nodorum blotch, caused by Leptosphaeria nodorum, and to tan spot, caused by Pyrenophora tritici-repentis, in wheat.

Cultivars from different countries were screened for resistance to these diseases in seedling tests conducted under controlled environmental conditions. The proportion of resistant cultivars identified was low; 12% of 482 cultivars tested were resistant to septoria nodorum blotch and only 2% of 350 cultivars tested were resistant to tan spot. Resistance to septoria nodorum blotch was found also in species closely related to wheat. Cultivars 7990-244A, 81IWWMN 2095, Erik, Hadden and ZG 7581-83 were resistant to both diseases. Most resistant cultivars were winter wheats and the level of resistance in spring wheats was low. An important exception is spring wheat cultivar 86ISMN 2137 which was highly resistant. All currently registered western Canadian wheat varieties were susceptible to both diseases.

The genetic control of resistance was studied in seedling tests using crosses between resistant and susceptible parents. Parents resistant to septoria nodorum blotch were 71-10-12, 81IWWMN 2095, 82IWWMN 2019, 83IWWMN 2051, Coker 76-35, EA 13, EE 8, Hadden, Missouri Queen, Moking, Red Chief and ZG 7581-83. Resistance was controlled by a single gene in all crosses and evidence for at least two different genes for resistance was obtained. Cultivars 81IWWMN 2095, Hadden and ZG 7581-83 were found to carry single genes for resistance to tan spot also.

Race formation in L. nodorum was studied in controlled environment tests by inoculating a set of cultivars with a range of isolates collected from different areas in Saskatchewan. A significant isolate x cultivar interaction was detected but appeared due mainly to the aberrant behaviour of one cultivar. While differences in the pathogenicity of isolates were measured, no clear evidence for the existence of physiologic races of this pathogen were found. Cultivar 81IWWMN 2095 was highly resistant to all isolates used whereas other cultivars varied in their level of resistance.

Field tests were conducted in 1988 and 1989 at Saskatoon and Weirdale to study the association of resistance with agronomic traits and to relate seedling and adult plant disease reaction. Despite artificial inoculation and irrigation, disease epidemics could not be established at Saskatoon. However, a low level of disease developed from natural infection at Weirdale and disease rating was possible. A good relationship was found between field and seedling ratings, thus confirming the validity of using seedling testing to study and breed for resistance. Resistance tended to be associated with tallness and late maturity, but not with growth habit.

These diseases are major diseases of wheat in Saskatchewan and have increased in occurrence in recent years due largely to more continuous cropping and reduced tillage operations. In north-central regions yield losses attributed to these diseases are 14 - 15%, even at moderate disease levels. Development and use of resistant varieties will minimize these losses, thus enhancing the competitiveness of Saskatchewan wheat producers, and also provide an environmentally safe method of control.

The results of this project provide the basis for such development. The resistance sources, simple genetic control and good correspondence between seedling test and field reaction are factors which will facilitate breeding for resistance, a program which this project has already commenced. This study also is the first to demonstrate definitely single-gene control of resistance to L. nodorum and thus represents an important scientific contribution in this area.

1.0 INTRODUCTION

In recent years, the leaf spot disease complex of wheat has become increasingly important in Saskatchewan. The fungi, Leptosphaeria nodorum Muller, causing septoria nodorum blotch (glume blotch), and Pyrenophora tritici-repentis (Died.) Drechs., causing tan spot, are important members of this complex. These pathogens are widespread but of varying importance throughout Saskatchewan. L. nodorum is the major pathogen of the complex in the northern cropping areas whereas P. tritici-repentis seems more important in southern Saskatchewan.

In tests conducted in north-central Saskatchewan from 1984 to 1988, yield loss at moderate levels of disease averaged 14%, indicating the importance of this complex as a disease of wheat. By comparison, the estimated average annual yield loss caused by common root rot in Saskatchewan is 7%.

Host resistance and cultural practices are presently the only economical means of control and should be used together for the most effective results. Host resistance has been reported for both pathogens. It is generally described as quantitative and under complex genetic control, although monogenic control of resistance to P. tritici-repentis has been reported. Selection for resistance has been effective, indicating that development of resistant varieties is feasible. Therefore, this project was undertaken to provide the genetic information needed by plant breeders to produce resistant varieties. The objectives of the project were

(a) to identify sources of host resistance,

(b) to determine the genetic control of resistance and

(c) to examine the extent of variability for pathogenicity of these pathogens in Saskatchewan.

2.0 IDENTIFICATION OF SOURCES OF RESISTANCE

2.1 Materials and Methods

The hexaploid wheat cultivars tested for seedling resistance originated from many countries, including Australia, Canada, United States, USSR, Chile, Yugoslavia, Nepal, Ethiopia, France, Argentine, Brazil, Paraguay, Uraguay and China. They were obtained from the Department of Crop Science and Plant Ecology, University of Saskatchewan, Saskatoon, Agriculture Canada Research Stations at Winnipeg and Charlottetown, and the USDA International Septoria Nursery distributed by Dr. A.L. Scharen, Montana State University, Boseman, Montana. A total of 482 cultivars were tested for resistance to L. nodorum and 350 cultivars were tested for resistance to P. tritici-repentis.

In addition, 42 accessions of T. monococcum, 18 accessions of T. tauschii, 17 accessions of T. timopheevii and 32 accessions of T. dicoccum were tested for seedling resistance to L. nodorum. These were supplied by Dr. A. Limin, Crop Development Centre, University of Saskatchewan, Saskatoon.

Each seedling test was grown as a completely randomized design with two replications, each treatment replication consisting of six plants. The cultivar Kenyon was included as the susceptible control in each test. Inoculum was prepared from 14-day-old cultures of L. nodorum, grown on 15% V-8 juice agar (150 mL Campbell's V-8 juice, 1.5 g calcium carbonate, 15 g agar and 1000 mL water) at room temperature (ca. 22°C) under continuous fluorescent light, or from 10-day-old cultures of P. tritici-repentis cultured in an incubator on 15% V-8 juice agar at 21°C with a 12 h photoperiod. Best sporulation was obtained for L. nodorum if plates were placed in an upright position, but in an inverted position for P. tritici-repentis. The isolates of each pathogen were derived from single pycnidia isolated from diseased leaves collected at Kelvington, Saskatchewan. The inoculum was adjusted to an average spore concentration of 1.8×10^6 spores/mL and 2.7×10^3 spores/mL for L. nodorum and P. tritici-repentis, respectively. Two drops of the surfactant Tween 20 were added to each 100 mL of inoculum.

Seedling plants were inoculated at the 3-leaf stage by spraying them until run-off with the spore suspension and placed in a moisture chamber for 48 h. The third leaf was rated for disease reaction seven days after inoculation. Initially, a four-class rating scale was used, resistant, moderately resistant, moderately susceptible and susceptible. A resistant reaction was characterized by small, brown lesions with little or no necrosis or chlorosis. A susceptible reaction appeared as large necrotic lesions with extensive chlorosis. Later, a rating scale of 0 to 9 was used to assess disease reaction. A rating of 0 described a disease reaction of green leaves with pin-point brown lesions and a rating of 9 described completely chlorotic leaves with large necrotic lesions. Most cultivars rated as resistant were tested at least twice to confirm their reaction.

2.2 Results and Discussion

This screening method was effective in identifying seedling resistance to both pathogens. Fifty nine of 482 cultivars tested were resistant to *L. nodorum* in the seedling stage (Table 1). A cultivar was considered resistant if rated as resistant on the 4-class rating scale or if its mean rating was less than 2.0 on the 0-9 scale. Most of these were winter wheats (85%) and, in general, the highest level of resistance was found in winter wheat cultivars. One important exception was cultivar 86ISMN 2137, a spring wheat with a high level of resistance (rating = 0.1). Because use of this line will avoid the vernalization needed when working with winter wheats, it will be a valuable source of resistance for breeding programs.

Resistance was found also in the other Triticum species tested (Table 2). *T. dicoccum* was the poorest source of resistance. Only 13% of *T. dicoccum* accessions were rated resistant compared to 50%, 47% and 36% of *T. tauschii*, *T. timopheevii* and *T. monococcum* accessions, respectively. Based on resistance level and crossability with hexaploid wheat, accessions PI 289599 (*T. monococcum*) and PI 290518 (*T. timopheevii*) were identified as the most useful sources.

Table 1. Sources of resistance to Leptosphaeria nodorum and to Pyrenophora tritici-repentis identified in growth room seedling tests

Lines resistant to L. nodorum

71-10-12	Baldrock	Pat 72180
7990-244A*	Bladette de Puylaurens	PI 341604
81IWWMN 2083	Carifen	PI 436492
81IWWMN 2093	Coker 76-35	Poso
81IWWMN 2094	Defiance	Red Chief
81IWWMN 2095*	EA 13	SPR 84-3
82IWWMN 2019	EE 1	WTR 71-8ST
82IWWMN 2057	EE 8	WTR 75-1ST
83IWWMN 2045	Erik*	WTR 81-12SN
83IWWMN 2051	Fat Bugday	WTR 81-17ST
85ISMN 2039	Fleche d'or	WTR 81-20SN
86ISMN 2122	Hadden*	WTR 81-39ST
86ISMN 2127	Harvest Queen	WTR 83-17
86ISMN 2137	Hybrid 143	ZG 991-85
86IWMN-57ST	JCR 655	ZG 3021-84
86IWMN-75SN	Lacos III 262	ZG 6910-82
86IWMN-92SN	Missouri Queen	ZG 7581-83*
86IWMN-111SN	Moking	ZG 8085-83
86WRWWN-21SN	Nugaines	Yamhill
86WRWWN-25SN	Oasis	

Lines resistant to P. tritici-repentis

7990-244A*	Erik*	
81IWWMN 2095*	Hadden*	NS 18-77
Ble Blanc de la Reole	Nanjing 7317	ZG 7581/83*

* Lines resistant to both pathogens.

Table 2. Sources of resistance to Leptosphaeria nodorum identified in Triticum species in growth room seedling tests

T. monococcum	T. tauschii	T. timopheevii
PI 167591	CI 5	PI 119442
PI 167627	CI 16	PI 289752
PI 190942	CI 18	PI 290518
PI 221329	CI 52	PI 341602
PI 221393	RL 5523	CI 11802
PI 254195	RL 5534-3	CI 14133
PI 272562	RL 5536-4	
PI 277140	RL 5548	
PI 286068	RL 5561	
PI 289599		
PI 289605		
PI 355529		
PI 355546		
CI 13963		
CI 14090		

Considerably fewer cultivars resistant to P. tritici-repentis were identified. Only eight of the 350 tested showed resistance. However, five were resistant to both L. nodorum and P. tritici-repentis. These five cultivars were 7990-244A, 81IWWMN 2095, Erik, Hadden and ZG-7581/83. It is not known whether a relationship exists in these cultivars between the genes for resistance to the two pathogens.

Fifteen registered Canadian cultivars, spring wheats Benito, Columbus, Conway, Glenlea, HY 320, Katepwa, Kenyon, Neepawa, Oslo, Park, Roblin, winter wheats Norstar and Norwin, and durum wheats Sceptre and Wakooma were tested and found to be susceptible to both pathogens.

Results from the USDA International Septoria Nurseries identified cultivars 81IWWMN 2083, 81IWWMN 2093, 81IWWMN 2095 and 82 IWWMN 2019 as possessing good resistance to L. nodorum in field tests conducted in 11 countries. These same cultivars were identified in this project as sources of resistance on the basis of their seedling reaction. This suggests good correspondence between seedling and field reaction.

3.0 GENETIC CONTROL AND HERITABILITY OF RESISTANCE

3.1 Materials and Methods

Twelve sources of resistance and the susceptible cultivar Kenyon were used in this study. The resistant cultivars were 71-10-12, 81IWMMN 2095, 82IWMMN 2019, 83IWMMN 2051, Coker 76-35, EA 13, EE 8, Hadden, Missouri Queen, Moking, Red Chief and ZG7581-83. All are winter wheats and required vernalization before crosses and backcrosses could be made to Kenyon. Vernalization was achieved by growing plants at 5-8°C for 6 weeks, commencing at the 3-leaf stage, and then at 15°C for 1 week.

The resistant cultivars were crossed with Kenyon and some F1 plants backcrossed to Kenyon (BC1), while others were selfed to produce F2 seed. Backcross F2 families were produced by selfing backcrossed plants and F3 families by selfing randomly chosen F2 plants. Approximately 25 seedlings of each backcross F2 family or F3 family were tested.

All tests were conducted on seedling plants in a controlled environment growth room with a 16 h photoperiod and 22°C day/16°C night temperatures. Inoculum production and inoculation procedures were as described in Section 2.1. Spore concentration of the inoculum averaged 1.2 x 10^6 spores/mL. F2 populations, and all backcross F2 and F3 families were tested with the Kelvington isolate of L. nodorum. In addition, the same backcross F2 families of some crosses were tested with the Weirdale isolate. The parents and F1 generation of the appropriate cross were included in each test. The third leaf of each plant was rated on the 0-9 rating scale and a plant was classed as resistant if its rating did not fall within the range of the F1 generation and the susceptible parent. The chi-square test was used to test for goodness-of-fit of the observed segregation ratios to the expected Mendelian segregation ratios.

Cultivars 81IWMMN 2095, Hadden and ZG 7581-83 were studied in crosses with susceptible cultivar Kenyon to determine the inheritance of resistance to P. tritici-repentis. Parental, F1, F2 and backcross F2 or F3 generations

were tested with the Kelvington isolate of P. tritici-repentis using the methods described for the L. nodorum study. The inoculum concentration used averaged 4.4 x 10^3 spores/mL.

3.2 Results and Discussion

3.2.1 Frequency Distributions

3.2.1.1 Leptosphaeria nodorum

No overlap occurred between the parental ranges and with the exception of Red Chief, all resistant parents were rated in the 0-2 range (Table 3). These ratings indicated that Red Chief possessed the lowest level of resistance, but there was little difference in resistance among the other resistant parents. All F1 distributions fell at the susceptible end of the rating scale. The F1 distributions of crosses 71-10-12 x Kenyon and EA 13 x Kenyon fell completely within the susceptible parental range, expected if resistance was completely recessive. In the other crosses, the F1 range was only partially included in the susceptible range and indicated that resistance was partially recessive. However, since the number of F1 and parental plants tested was small in most cases, conclusions on the degrees of recessiveness of resistance in these crosses are tentative. All F2 generation means were lower than the F1 means, suggesting this possible bias. In the 83IWWMN 2051, Coker 76-35 and EE8 crosses the F2 means were similar to the midparental value. This suggested dominance was absent in these crosses.

The F2 distributions were bimodal suggesting segregation of a single gene pair. The discontinuity in the frequency distribution usually corresponded to the resistant end of the F1 range, indicating it could be used as the breakpoint between resistant and susceptible classes. The discontinuity was least evident in the F2 distribution of the Coker 76-35 x Kenyon cross due to a high number of plants with intermediate ratings. This would be expected if no dominance was present. Consequently there was uncertainity as to where the division between resistant and susceptible classes should be made.

Table 3. Frequency distributions of individual plant disease ratings in the parental, F1 and F2 generations of resistant x susceptible crosses tested with Leptosphaeria nodorum in growth room seedling tests

Generation	Number of plants in disease rating class										Total	Mean	S.D.
	0	1	2	3	4	5	6	7	8	9			
71-10-12 x Kenyon													
71-10-12	8	6									14	0.4	0.51
F1							8		4		12	6.7	0.99
F2	11	43	26	24	6	112	70	57	62	3	414	5.1	2.31
Kenyon							1	1	6	5	13	8.2	0.90
81IWWMN 2095 x Kenyon													
2095	8	9	8								25	1.0	0.82
F1						10	5	8	3		26	6.2	1.08
F2	18	50	47	11	28	142	114	103	94		607	5.2	2.26
Kenyon								1	11	11	23	8.4	0.59
82IWWMN 2019 x Kenyon													
2019	1	2									3	0.7	0.58
F1						2	3	3	7		15	7.0	1.13
F2		19	29	48	13	80	74	94	147	2	506	5.9	2.08
Kenyon								3	13		16	7.8	0.40
83IWWMN 2051 x Kenyon													
2051	1	3	1								5	1.0	0.71
F1						3	1				4	5.2	0.50
F2	63	141	69	22	6	684	67	32	20		1104	4.1	1.93
Kenyon									2	1	3	7.3	0.58
Coker 76-35 x Kenyon													
Coker	2	3									5	0.6	0.55
F1						2	2	1			5	5.8	0.84
F2	84	47	58	50	506	136	49	31			961	4.7	1.63
Kenyon								1	3		4	7.8	0.50

Table 3. (cont.)

Generation	Number of plants in disease rating class										Total	Mean	S.D.
	0	1	2	3	4	5	6	7	8	9			
EA 13 x Kenyon													
EA 13	1	3	1								5	1.0	0.71
F1						2	2	1			5	5.8	0.84
F2		83	84	60	24	322	140	99	126	16	954	5.1	2.12
Kenyon									2	1	5	7.2	1.64
EE8 x Kenyon													
EE8	2	2									4	0.5	0.58
F1						2	3				5	5.6	0.55
F2	21	160	30	8	13	495	106	44	48		925	4.4	2.03
Kenyon							2		3		5	7.2	1.10
Missouri Queen x Kenyon													
MQ	3	9	3								15	1.0	0.66
F1						5	4	4	1		14	6.1	1.00
F2	18	75	35	4	33	176	121	104	79	4	649	5.1	2.26
Kenyon								4	8	3	15	7.9	0.70
Moking x Kenyon													
Moking	9	1	4								14	0.6	0.93
F1						4	3	4	2		13	6.3	1.11
F2	10	46	64	1	65	133	108	130	236	11	804	5.8	2.25
Kenyon							1	1	8	1	11	7.8	0.75
Red Chief x Kenyon													
RC	4	1	5	1	3						14	1.9	1.51
F1								2	10	3	15	8.1	0.59
F2	7	17	48	2	104	45	53	79	310	100	765	6.6	2.26
Kenyon									5	6	11	8.5	0.52

3.2.1.2 Pyrenophora tritici-repentis

In general, the expression of resistance to this pathogen was lower than was observed for L. nodorum. Hadden (rating 1.0) was the most resistant of the three cultivars tested (Table 4). No overlap occurred between the distribution of the resistant parent and that of either the F1 generation or the susceptible parent. These last two distributions overlapped indicating that resistance was recessive. However in all crosses except ZG 7581-83 x Kenyon, the F1 rating mean was significantly different from the mean of the susceptible parent. This suggested that resistance was completely recessive in that cross, but only partially recessive in the others.

The F2 distributions were bimodal and, as observed earlier, the discontinuity usually coincided with the resistant end of the F1 generation range (Table 4). Thus, plants rating 4 or lower were classed as resistant in the 81IWWMN 2095 and Hadden cross populations. In the ZG 7581-83 cross, which exhibited the lowest level of resistance, the F2 discontinuity occurred at rating class 5 and classification of resistant and susceptible plants was adjusted accordingly.

3.2.2 Mendelian Analysis

3.2.2.1 Leptosphaeria nodorum

The F2 population segregation data of each cross fitted the 1 resistant : 3 susceptible ratio expected if a single gene controlled resistance and was recessive in effect (Table 5). However, an alternate hypothesis was possible for the Coker 76-35 cross. If F2 plants rating 4 or less were classed as resistant, instead of using the breakpoint at rating as suggested by the discontinuity in the distribution, a 745 R : 216 S segregation would be obtained. This fitted a 3:1 ratio (P=0.05-0.10) expected if a single dominant gene for resistance was segregating.

Data from tests of F2 backcross families and F3 families (Table 6) confirmed the conclusion of a single gene controlling resistance in seven resistant cultivars. F2 backcross family segregation data fitted the

Table 4. Frequency distributions of individual plant disease ratings in the parental, F1 and F2 generations of resistant x susceptible crosses tested with Pyrenophora tritici-repentis in growth room seedling tests

Generation	Number of plants in disease rating class										Total	Mean	S.D.
	0	1	2	3	4	5	6	7	8	9			
81IWMMN 2095 x Kenyon													
2095			10	4							14	2.3	0.47
F1						8	2	2	2		14	5.9	1.17
F2			70	56	35	209	104	64	37		575	5.0	1.66
Kenyon								2	12		14	7.9	0.36
Hadden x Kenyon													
Hadden		4									4	1.0	0.0
F1						6	1	3			10	5.7	0.95
F2		33	35	18	18	129	80	103	47		463	5.3	1.98
Kenyon							1	2	7		10	7.6	0.70
ZG 7581-83 x Kenyon													
ZG				5							5	3.0	0.0
F1								1	4		5	7.8	0.45
F2			5	42	179	35	57	134	447		1002	6.8	1.85
Kenyon									3	2	15	8.4	0.55

Table 5. Segregation for reaction to isolates of Leptosphaeria nodorum in F2 populations of resistant x susceptible crosses in growth room seedling tests

Resistant parent	Isolate	Number of plants			Ratio expected	P value
		Res	Susc	Total		
71-10-12	Kelv*	110	304	414	1:3	0.25-0.50
81IWMMN 2095	Weir	154	453	607	1:3	0.75-0.90
82IWMMN 2019	Weir	109	397	506	1:3	0.05-0.10
83IWMMN 2051	Kelv	301	803	1104	1:3	0.05-0.10
Coker 76-35	Kelv	239	722	961	1:3	0.90-0.95
EA 13	Kelv	251	703	954	1:3	0.25-0.50
EE 8	Kelv	232	693	925	1:3	0.90-0.95
Hadden	Kelv	92	262	354	1:3	0.50-0.75
Hadden	Weir	140	416	556	1:3	0.90-0.95
Missouri Queen	Kelv	165	484	649	1:3	0.75-0.90
Moking	Kelv	186	618	804	1:3	0.10-0.25
Red Chief	Kelv	171	594	765	1:3	0.05-0.10
ZG 7581-83	Kelv	72	201	273	1:3	0.25-0.50
ZG 7581-83	Weir	104	324	428	1:3	0.50-0.75

* Kelv = Kelvington isolate, Weir = Weirdale isolate of L. nodorum.

Table 6. Segregation for reaction to isolates of Leptosphaeria nodorum of backcross F2 families and F3 families of resistant x susceptible crosses in growth room seedling tests

Resistant Parent	Isolate	Number of families				Ratio expected	P Value
		Res	Seg	Susc	Total		
81 IWWMN 2095	Kelv		40	54	94	1:1	0.10-0.25
	Weir		43	51	94	1:1	0.25-0.50
82 IWWMN 2019	Kelv		36	50	86	1:1	0.10-0.25
	Weir		38	46	84	1:1	0.25-0.50
Coker 76-35	Kelv		29	32	61	1:1	0.50-0.75
	Weir		30	31	61	1:1	0.75-0.90
EA 13	Kelv		51	41	92	1:1	0.25-0.50
	Weir		46	47	93	1:1	0.90-0.95
Red Chief	Kelv		31	35	36	1:1	0.50-0.75
	Weir		35	31	66	1:1	0.50-0.75
Hadden	Kelv	6	26	11	43	1:2:1	0.10-0.25
ZG 7581-83	Kelv	12	24	19	55	1:2:1	0.25-0.50

expected 1 segregating : 1 susceptible family ratio when tested with two fungal isolates. All segregating F2 backcross families segregated 1 resistant : 3 susceptible (data not shown) and the individual segregation of F2 backcross plants, summed for all segregating families fitted the expected 1:3 ratio in most cases. The three instances where a 1:3 segregation ratio did not fit were considered to result from bias introduced by the small number of plants tested in some families. The observed data for the second isolate did fit the 1:3 ratio. In all cases, however, there was a lower than expected number of resistant plants. The F3 family segregation data likewise fitted the expected 1 resistant : 1 segregating : 1 susceptible family segregation for the two crosses tested (Table 7). The individual plant segregation data combined for all segregating families showed poor fit to the expected 1:3 ratio, due to an excess of resistant plants. This is most likely the result of uneven inoculation resulting in genetically susceptible plants being misclassified.

The F2 backcross families of five crosses were tested with two different isolates of L. nodorum. Similar results for family segregation were obtained for each isolate. This provides some evidence that the effect of the genes for resistance carried by those cultivars is not isolate-specific.

Random F3 families have been produced for eight resistant susceptible crosses. Testing of these families is in progress. This will provide the data necessary to confirm the F2 segregation data in the 71-10-12, 83IWMMN 2051, EE 8, Missouri Queen and Moking crosses, and additional data for the 81IWMMN 2095, Coker 76-35 and Red Chief crosses.

Additional F2 segregation data for resistance to the Kelvington isolate was obtained during production of the above F3 families (Table 8). These data supported previous results except for control of resistance in Coker 76-35 and 71- 10-12 where single-gene control was still indicated but resistance appeared to be dominant. Although genetic control is not in question, additional studies are needed to resolve the conflicting results for the type of dominance operating.

Table 7. Segregation of plants for reaction to two isolates of *Leptosphaeria nodorum* in segregating backcross F2 families of five resistant x susceptible crosses tested in growth room seedling tests.

Resistant parent	Isolate	Number of plants			Ratio expected	P value
		Res	Susc	Total		
81 IWWMN 2095	Kelv	304	1010	1314	1:3	0.1 -0.25
	Weir	240	985	1225	1:3	<0.01
82 IWWMN 2019	Kelv	194	587	781	1:3	0.90-0.95
	Weir	181	679	869	1:3	<0.01
Coker 76-35	Kelv	145	507	652	1:3	0.1 -0.25
	Weir	149	489	638	1:3	0.25-0.50
EA 13	Kelv	271	869	1140	1:3	0.25-0.50
	Weir	233	799	1032	1:3	0.05-0.10
Red Chief	Kelv	142	610	752	1:3	<0.01
	Weir	177	563	740	1:3	0.25-0.50

Table 8. Segregation for reaction to Leptosphaeria nodorum in F2 populations of resistant x susceptible crosses in growth room seedling tests

Cross	Number of F2 plants Res	Susc	P value (1:3)	P value (3:1)
EE8 x Kenyon	24	104	0.10-0.25	
Missouri Queen x Kenyon	22	49	0.10-0.25	
Moking x Kenyon	29	49	0.01-0.05	
Red Chief x Kenyon	11	46	0.25-0.50	
81IWWMN 2095 x Kenyon	13	47	0.75-0.90	
83IWWMN 2051 x Kenyon	36	91	0.25-0.50	
Coker 76-35 x Kenyon	40	20	< 0.005	0.10-0.25
71-10-12 x Kenyon	105	48	< 0.005	0.05-0.10

Table 9. Segregation for reaction to Pyrenophora tritici-repentis in F2 populations of resistant x susceptible crosses in growth room seedling tests

Resistant parent	Number of plants Res	Susc	Total	Ratio expected	P value
81IWWMN 2095	161	414	575	1:3	0.05-0.10
Hadden	104	359	463	1:3	0.10-0.25
ZG 7581-83	261	741	1002	1:3	0.75-0.90

Table 10. Segregation for reaction to Pyrenophora tritici-repentis of backcross F2 families and F3 families of resistant x susceptible crosses in growth room seedling tests

Resistant parent	Number of families Res	Seg	Susc	Total	Ratio expected	P value
81IWWMN 2095		47	41	88	1:1	0.50-0.75
Hadden	11	23	12	46	1:2:1	0.75-0.90
ZG 7581-83	10	27	21	58	1:2:1	0.50-0.10

3.2.2.2 Pyrenophora tritici-repentis

Segregation data for the F2 generation is shown in Table 9 and for the backcross F2 families and F3 families in Table 10. All observed segregations fitted the ratios expected for single gene control of resistance. The lower level of resistance noted in the ZG 7581-83 cross suggested the gene for resistance possessed by this cultivar is different to that carried by each of the other cultivars.

3.2.3 Estimates of Gene Number

Estimates of gene number in the crosses studied suggested one or two genes controlled resistance to each pathogen in all crosses except Coker 76-35 x Kenyon (Tables 11, 12). In this cross there was a clustering of plants around intermediate rating values resulting in a reduced estimate of genetic variance and a higher gene estimate. Since the Castle-Wright formula used to obtain these estimates does not account for the presence of dominance, these must be considered minimal estimates. Like the Mendelian analysis, simple gene control of resistance is indicated.

3.2.4 Heritability

The variation in disease reaction observed in the non-segregating parental and F1 generations tested in the inheritance studies indicated disease reaction was subject to environmental effects. To determine the magnitude of these effects, heritability estimates for seedling resistance were obtained by calculating broad-sense heritability estimates by variance component analysis and narrow-sense estimates by regression of F4 family means on F3 family means.

3.2.4.1 Leptosphaeria nodorum

Broad-sense heritability estimates were similar for all crosses and ranged from 0.71 to 0.90 (Table 11). The narrow-sense estimates were lower since they do not include gene effects due to dominance and were estimated

Table 11. Estimates of broad and narrow-sense heritability of resistance to Leptosphaeria nodorum in growth room seedling tests

Cross	Broad-sense heritability[a]	Narrow-sense heritability[b]	Gene number[c]
71-10-12 x Kenyon	0.87		1.6
81IWWMN 2095 x Kenyon	0.86	0.63	1.6
82IWWMN 2019 x Kenyon	0.86		1.7
83IWWMN 2051 x Kenyon	0.90		1.5
Coker 76-35 x Kenyon	0.84		2.9
EE 8 x Kenyon	0.85		1.5
EA 13 x Kenyon	0.71		1.6
Moking x Kenyon	0.83		1.5
Missouri Queen x Kenyon	0.88		1.3
Red Chief x Kenyon	0.81		1.3
ZG 75811-83 x Kenyon		0.53	

[a] Heritability = (VF2 - (VP1 + VF1 + VP2)/3)/VF2.
[b] Heritability calculated as the regression coefficient when F4 family means are regressed on F3 family means.
[c] Estimated from the F2 generation using the Castle-Wright formula.

Table 12. Estimates of broad-sense heritability of resistance to Pyrenophora tritici-repentis in growth room seedling tests

Cross	Broad-sense heritability[a]	Gene number[b]
81IWWMN 2095 x Kenyon	0.79	1.8
Hadden x Kenyon	0.88	1.6
ZG 7581-83 x Kenyon	0.95	1.1

[a] Heritability = (VF2 - (VP1 + VF1 + VP2)/3)/VF2.
[b] Estimated from the F2 generation using the Castle-Wright formula.

from populations in which the level of homozygosity was higher.

These estimates indicate that resistance is moderately to highly heritable and that dominance is involved. Selection for resistance will be effective and since susceptibility is dominant, selection for resistance would be more effective than selection against susceptible individuals. Strictly speaking, these heritability estimates are valid only for growth-room seedling tests, however results from limited attempts to select for resistance in field nurseries suggest that resistance is moderately heritable under field conditions.

3.2.4.2 Pyrenophora tritici-repentis

Broad-sense heritability estimates ranged from 0.79 to 0.95, indicating that expression of resistance is highly heritable (Table 12). However, given the small numbers of plants tested in the parental and F1 generations, underestimation of the variances in these non-segregating generations and thus of the environmental variance is likely. Although the heritability estimates obtained are probably biased upwards, effective selection for resistance in seedling tests can be expected.

3.2.5 Allelism of Genes for Resistance to Leptosphaeria nodorum

Studies of F2 populations of crosses between resistant cultivars 82IWMMN 2019, 81IWMMN 2095 and Coker 76-35 suggested that 81IWMMN 2095 and 82IWMMN 2019 possess the same gene for resistance. The segregation ratio was 51 resistant : 0 susceptible plants in the cross involving those two cultivars. However, this gene appears to be different to that in Coker 76-35. The segregation ratios in the Coker crosses fitted the 13 resistant : 3 susceptible ratio expected if one dominant and one recessive gene were segregating, 82IWMMN 2019 x Coker = 40 Resistant : 10 susceptible (P=0.75-0.90), 81IWMMN 2095 x Coker = 18 Resistant : 6 Susceptible (P=0.25-0.50). The presence of a dominant gene for resistance in Coker 76-35 was suggested earlier.

Difficulty was encountered in producing the complete diallel set of crosses from the resistant parents. Synchronization of flowering to permit crosses to be made was very difficult because of the different vernalization requirements of these parents, all winter wheats. However, most crosses have now been made and F1 plants will be selfed and crossed with Kenyon this summer to produce the populations necessary to complete this aspect of the work. The resistance gene present in 81IWMMN 2095 and 82IWMMN 2019 is tentatively designated as *Ln1* and that present in Coker 76-35 as *LnC*.

3.2.6 Location of Genes for Resistance to Leptosphaeria nodorum

This preliminary study was undertaken to explore the feasibility of using biochemical markers to identify the presence of a resistance gene rather than disease reaction. The advantage of the biochemical marker is that the results are less subject to error than are disease tests. Two approaches have been undertaken. The first involved tests to determine whether an association exists between a known biochemical marker and resistance to L. nodorum. The second is conventional gene mapping using a monosomic series.

3.2.6.1 Use of biochemical markers to identify resistance genes

Gel electrophoresis was used to determine whether resistance to L. nodorum in T. timopheevii was associated with a biochemical marker. Banding patterns for each isozyme studied were produced for T. timopheevii accession PI 290518, a resistant Wakooma x T. timopheevii BC1F6 line and susceptible durum cultivars Sceptre and Wakooma using the appropriate extraction and staining procedure for each enzyme. The isozyme markers studied and their chromosome group location were Acph (4S), Aco (5L, 6L), Amp (4S), Ep (7L), Glu (1L,1S), Mdh (5S), Pgm (4S), Skdh (5S) and Tpi (5l).

Some differences in banding patterns were observed, particulaly for Aco in Sceptre and the resistant BC1F6 line, but no specific association with resistance was noted. It was concluded that the chromosomal location of resistance needs to be determined to identify potentially useful biochemical markers.

3.2.6.2 Location of genes for resistance to L. nodorum by monosomic analysis

Verification of the identity of lines monosomic for each of the A genome chromosomes of wheat by ditelosomic analysis has been completed. This monosomic series will be used to determine the chromosomal location of the host resistance genes identified in the genetic analysis. Successful chromosome location of the resistance genes will not only identify those biochemical markers which can be used to tag those genes but also will indicate whether useful DNA markers (RFLPs) exist. Application of molecular genetics technology would greatly aid this disease resistance program.

Monosomic plants are identified by cytological examination and crossed with a resistant cultivar to determine chromosomal location of the resistant gene or with Chinese Spring to maintain the line. Since the genes under study have recessive effects, F1 plants of the critical cross will segregate for resistance whereas all F1 plants of non-critical crosses will be susceptible. Location of the genes in the A genome chromosomes is being tested first since data obtained during the transfer of resistance from T. timopheevii suggested that the resistance gene(s) were located on the A genome.

4.0 FIELD STUDIES

These studies were conducted to study disease progress in resistant spring wheat cultivars, to examine the relationship between resistance and agronomic traits and to determine the relationship between seedling and adult plant reaction.

4.1 Material and Methods

Field disease nurseries were grown in 1988 and 1989 at the North Seed Farm, University of Saskatchewan, Saskatoon and at Weirdale in north-central Saskatchewan on the farm of Mr. J. Meyer. The Saskatoon nurseries were seeded into wheat stubble in both years. The Weirdale nursery was seeded into pea stubble in 1988 and into canola stubble in 1989; both sites had not been seeded into wheat for two years.

4.1.1 1988 Season

F3 families derived from random F2 plants with a spring growth habit from crosses 81IWWMN 2095 x Kenyon, Hadden x Kenyon and ZG 775811-83 were grown at both locations. For each cross 50 F3 families were seeded in a 3-replicate randomized complete block design. Each replication also included one row of the winter wheat resistant parent and three rows of Kenyon. Plot size was 1 row x 4 m, with rows seeded 30 cm apart. The nursery at Saskatoon was seeded on May 21, 1988 and at Weirdale on May 28, 1988. Fertilizer was applied at seeding according to soil test recommendations.

Inoculation of the Saskatoon nursery was attempted by spreading infected wheat straw between the rows when plants were on the 3-4 leaf stage and spraying the plants with a spore suspension of L. nodorum on two occasions, at the 5-leaf stage and again at the flag leaf stage. After each inoculation, the plots were irrigated intermittently during the night for three nights. Subsequently, nightly irrigation was repeated twice each week.

At Weirdale natural infection was relied upon for disease development. The growth habit, height and relative growth stage (Zadok's scale) at three

times during the season were recorded. The last was used as a measure of relative maturity.

4.1.2 1990 Season

In 1989 the Weirdale nursery contained unreplicated 2 m rows of F3 families of crosses 82IWWMN 2019 x Kenyon, Red Chief x Kenyon, EA 13 x Kenyon, Coker 76-35 x Kenyon, 83 IWWMN 2095 x Kenyon, EE 8 x Kenyon, Moking x Kenyon and F4 families of crosses 81IWWMN 2095 x Kenyon, ZG 7581-83 x Kenyon and Hadden x Kenyon. The Saskatoon nursery was a duplicate of the Weirdale nursery and also contained F5 families derived from the 81IWWMN 2095 x Kenyon F3 families grown at Weirdale in 1988. At both locations a row of Kenyon was seeded every fifth row. Seeding dates were May 29, 1989 at Weirdale and June 2, 1989 at Saskatoon. Inoculation and irrigation procedures were the same as in 1988 except that only the Kenyon rows were inoculated with the spore suspension.

4.2 Results and Discussion

The hot, dry weather encountered in both years prevented adequate disease development at Saskatoon and slowed it at Weirdale. The Saskatoon nursery served only as a source of seed in each year.

Sufficient disease developed late in the season at Weirdale to permit tentative disease ratings of the lines in the nursery. These ratings, based on a four-class qualitative scale (R, MR, MS and S), were adequate for comparisn of field and seedling ratings but not for quantitative analysis of the relationship between disesae reaction and the agronomic traits measured. Visual examination of the data, however, did suggest resistant lines tended to be taller and slower developing than susceptible ones. No relatioship appeared to exist between disease reaction and winter or spring growth habit. Disease development was too late and of insufficient severity to permit any study of disease progression.

4.2.1 Relationship between Seedling and Adult Plant Reaction

As most of this project involved testing of seedling plants, it was of interest to examine the relationship between disease reaction at the seedling and adult plant stage. Data to test this relationship was obtained in a number of different ways.

F1 and F2 plants with a spring growth habit of cross 81IWMMN 2095 x Kenyon were tested at both seedling and adult plant stage. Few differences in disease reaction, classed as resistant or susceptible, were found between the seedling and the adult plant stage. Similar results were obtained when random F2 plants and backcross F2 families of crosses involving Red Chief, 82 IWMMN 2019 and Coker 76-35 were tested. These results suggest that the gene for resistance in each of these cultivars conditions resistance to L. nodorum throughout the life of the plant.

Comparison of seedling and field reaction of F3 families and of seedling reaction of up to 10 F3-derived F4 families was made for two crosses grown in field disease nursery at Weirdale in 1988 (Table 13). Of 17 F3 families rated resistant or moderately resistant in the field, six were susceptible (rating 4.0) based on the mean family seedling reaction. However, F4 family seedling reaction indicated only three families were misclassified by their field reaction. One family (23), rated as very resistant in the field, was the most resistant in both seedling tests. Results for cross ZG 75811-83 x Kenyon were similar (data not shown). Material to conduct a similar study was harvested from the 1989 field nursery but seedling testing has not been completed.

These results indicate a good correspondence between seedling and adult plant reaction and thus support seedling testing as an appropriate method of screening for resistance. Resistant lines can be identified in the field although a low level of misclassification may occur. Since disease severity in the 1988 field nursery at Weirdale was low, the misclassification that occurred in that year was probably due to low disease pressure resulting in moderately susceptible plants appearing more resistant than they really were.

Table 13. Comparison of seedling and adult field disease reactions to L. nodorum reactions F3 and F4 plant of cross 81 IMMMN 2095 x Kenyon

F3 family designation	F3 seedling reaction[a]	F3 field reaction	Mean F4 seedling reaction[b]
18	5.5*	R-MR	3.8
19	5.8*	R-MR	5.1*
20	6.0*	R	6.3*
21	2.4	R	3.2
22	2.0	R	1.8
23	1.1	VR	1.4
26	3.5	R	4.0
31	5.3*	R-MR	3.3
34	6.5*	R	3.9
36	2.0	R	2.5
37	2.7	R	3.6
38	2.0	R	2.2
40	7.8*	R-MR	6.2*
41	3.2	R-MR	2.0
42	3.8	R	3.3
43	2.2	R	3.6
49	0.9	R	0.7

[a] Seedling reaction on 0-9 scale, 10 to 15 plants rated per family.
[b] Mean of 6 to 10 families (10-15 plants per family rated).
* Seedling reaction (resistant vs. susceptible) different from field reaction.

5.0 PATHOGENIC VARIATION IN LEPTOSPHAERIA NODORUM

5.1 Material and Methods

Two experiments were conducted. The first involved 16 cultivars tested with 11 different Saskatchewan isolates of L. nodorum. The cultivars represented different levels of resistance to the Kelvington isolate and included winter wheats, triticale DU75 and spring wheat Kenyon. Each isolate was tested on seedlings of the 16 cultivars grown in a randomized complete block design with three replications, six plants per treatment replication. The same growth, inoculation and disease rating procedures were used as in the genetic studies (Section 3.1). Each isolate test was conducted at different times and repeated twice. Every effort was made to ensure that the conditions of each test were the same. However, testing each isolate at a different time meant that in a combined analysis of variance isolate differences will be confounded with environmental differences between tests.

The second experiment was conducted in the same manner as the first, but only 13 cultivars and seven different isolates were used. Each isolate was run only once.

5.2 Results and Discussion

The combined analysis of variance for disease ratings in the first experiment indicated highly significant differences for isolates and cultivars (Table 14). The cultivar x isolate interaction was just reached significance and was very small relative to the main effects. The interaction appeared due largely to the differential reaction of cultivar ZG 7581-83 to the various isolates. It was resistant to the Kelvington isolate but moderately susceptible or susceptible to the others (Table 15). This reversal of reaction was unexpected since in the genetic studies it was resistant to the Weirdale isolate also (Table 5). No obvious reason is available for the reversal of reaction and, until this discrepancy is resolved, the data for this cultivar should be considered with caution.

Table 15. Seedling reaction of 16 genotypes to 11 Saskatchewan isolates of Leptosphaeria nodorum in growth room seedling tests

Genotype	Pathogen isolate designation[a]											Number of virulent isolates
	KEL	WEIR	AYL	OUT	SEM	HB	PP	CHL	MEO	NB	SHL	
71-10-12	R[b]	R	R	R	R	R	R	R	R	R	MR	0
81IWWMN 2095	R	R	R	R	R	R	R	R	R	R	R	0
82IWWMN 2019	R	R	R	R	R	MR	MR	MR	MR	MR	MR	0
83IWWMN 2051	R	R	R	R	R	R	MR	MR	MR	MR	MR	0
EA 13	R	R	R	R	MR	R	R	R	R	R	MR	0
Missouri Queen	R	MR	R	MR	R	MR	MR	MR	MR	MR	MR	0
Moking	R	MR	R	MR	MR	MR	MR	MR	MR	MR	MR	0
EE 8	R	MR	MR	R	R	MR	MR	MR	MR	MS	MR	1
Oasis	R	MR	R	MR	R	MR	MR	MR	R	MR	MS	1
Red Chief	R	R	MR	R	R	MR	MR	MR	MR	MS	MR	1
DU75	MR	MR	MR	MR	MS	MS	MR	MR	MR	MS	MS	4
Hadden	R	MR	MR	MR	R	MR	MS	MS	MS	MS	MS	5
JCR 6C5	R	MR	MS	MR	MR	MR	MR	MS	MS	MS	MS	5
Coker 76-35	R	MR	MR	MS	MS	MS	MS	MS	MS	MS	MS	8
ZG 7581-83	MR	MS	MS	S	MS	MS	S	S	S	S	S	10
Kenyon	S	S	S	S	S	S	S	S	S	S	S	11
Number of resistant hosts	15	14	13	13	12	12	12	11	11	8	9	

[a] Area isolate collected from: AYL = Aylsham, CHL = Choiceland, HB = Hudson Bay, KEL = Kelvington, MEO = Meota, NB = North Battleford, OUT = Outlook, PP = Porcupine Plain, SEM = Semans, SHL = Shellbrook, WEIR = Weirdale.

[b] R = resistant (rating 0-2), MR = moderately resistant = (rating 2-4), MS = moderately susceptible (rating 4-6), S = susceptible (rating >6).

Table 14. Combined analysis of variance for disease ratings of 16 cultivars tested with 11 isolates of Leptosphaeria nodorum in growth room seedling studies

Source of variation	d.f.	Mean square	F value
Isolates	10	32.136	21.73**
Cultivars	15	105.861	71.58**
Isolates x cultivars	150	1.479	1.22*
Pooled error	825	1.225	

*, ** Significant at P=0.05, P=0.01, respectively.

Other cultivars, JCR 655, Coker 76-35, Hadden and Oasis, showed differential reactions to the isolates, but not to the same degree as ZG 7581-83 (Table 15). For these cultivars, the range of reaction was commonly MR to MS except to the Kelvington isolate. Thus, it seemed the differential reactions were due more to differences in aggressiveness than to differences in virulence. Furthermore, the rankings of the cultivars for disease rating changed little from isolate to isolate.

Large differences were observed in the level of resistance of cultivars to the different isolates (Table 15). Seven cultivars rated R or MR to all isolates and 81IWMMN 2095 and 71-10-12 showed the highest levels of resistance. The isolates also differed greatly in their virulence, as measured by the number of MS and S reactions they caused. The Kelvington isolate was the least virulent, causing a susceptible reaction only on Kenyon. The most virulent isolates were the North Battleford and Shellbrook isolates which caused susceptible reactions on seven and six culrivars, respectively.

In the second experiment, cultivar reactions were classed as either resistant or susceptible. As in the first study, no strong evidence for physiologic specialization but the superior resistance of 81IWMMN 2095 and 71-10-12 was noted again (Table 16). Differences in virulence of the isolates were also evident.

Table 16. Seedling reaction of 13 wheat genotypes to 7 Saskatchewan isolates of Leptosphaeria nodorum in growth room tests

Genotype	Pathogen isolate designation[a]							Number of virulent isolates
	BLN	COD	MQ	PAD	PW	SOM	85-13	
81IWWMN2095	R[b]	R	R	R	R	R	R	0
71-10-12	R	R	R	R	R	R	R	0
EA 13	R	R	R	R	R	R	R	0
83IWWMN2051	R	R	R	S	R	R	R	1
82IWWMN2019	R	R	R	S	S	R	R	2
Missouri Queen	S	R	S	R	R	R	R	2
Hadden	R	R	R	S	S	R	R	2
Moking	R	R	R	S	S	R	R	2
EE 8	R	R	R	S	S	R	R	2
Red Chief	R	R	R	S	S	R	R	2
Coker 76-35	S	S	S	S	S	R	R	5
ZG 7581/83	S	S	S	S	S	R	S	6
Kenyon	S	S	S	S	S	S	S	7
Number of resistant hosts	9	10	9	4	5	12	11	

[a] BLN = Blaine Lake, COD = Codette, MQ = Marquis, PAD = Paddockwood, PW = Paddockwood, SOM = Somerset.
[b] R = resistant, S = susceptible.

These studies were inconclusive in showing the occurrence of true physiologic specialization, but they clearly demonstrated that differences in isolate pathogenicity (resulting primarily from differences in aggressiveness) exist among Saskatchewan isolates of L. nodorum. These differences place restrictions on the usefulness of certain sources of resistance and stress the need to test resistant lines with a range of isolates before choosing sources for resistance breeding. Using a weakly pathogenic strain as the Kelvington isolate in genetic studies will reveal the presence of all genes for resistance, but will not indicate the widespread value of these genes. Two very valuable sources of resistance have been identified and either should form the basis of a resistance breeding program.

Further studies in this area are desirable to provide a more definitive answer to the question of whether physiologic specialization exists in this pathogen in Saskatchewan and to quantify the differences in pathogenicity.

6.0 RESISTANCE BREEDING

Resistance to L. nodorum from alien germplasm sources and from selected winter wheat sources is being transferred into genetic backgrounds adapted to Saskatchewan conditions by the backcross breeding procedure. In addition, homozygous resistant F4 and F5 families with a spring wheat growth habit have been developed by recurring selection for seedling resistance and selfing. These lines are attempts to obtain a series of homozygous spring wheat lines each carrying the resistance of the different winter wheat parents used in the genetic studies. In these forms, the resistances identified will be more easily used in breeding programs and in field studies.

Extraction of such lines for the resistant cultivars 81IWWMN 2095, Hadden and ZG 7581-83 is most advanced with F5 lines now available. F4 lines with resistance from 71-10-12, 83IWWMN 2051, EE8, Missouri Queen and Moking are currently being produced.

6.1 Transfer of Resistance from Hexaploid Wheat Sources

Resistance from 81IWWMN 2095 and Hadden is being transferred by backcrossing into spring wheat cultivar Kenyon. Material with 81IWWMN 2095 resistance is in the BC4 generation, ((81IWWMN 2095/2*Kenyon)F2//2*Roblin)F2 /3/Kenyon, and material with Hadden resistance is in the BC2 generation, (Hadden/Roblin)F2//2*Kenyon. An adequate level of resistance has been maintained throughout the backcrossing, but no plants as resistant as the resistant parent have been recovered. This suggests that expression of resistance is influenced by modifier genes.

6.2 Transfer of Resistance from Alien Sources

Resistance has been successfully transferred from T. timopheevii (accession PI 290518) into the durum wheat cultivar Wakooma. Two fully-fertile resistant BC1F6 lines of the cross Wakooma x T. timopheevii have been recovered. This provides a source of resistance for incorporation

into current durum wheat cultivars and a backcross program with susceptible cultivar Sceptre has been commenced.

Resistance is being transferred from resistant triticale DU75 into a hexaploid wheat background. DU75 is also highly resistant to septoria tritici blotch. A few BC1F1 seeds of cross DU75/2*Kenyon have been obtained. Crosses have also been made between DU75 and a *ph* mutant Chinese Spring stock. F1 seedlings from this cross have tested and all were moderately susceptible, indicating the resistant gene(s) have a recessive effect.

7.0 CONCLUSIONS

This study was undertaken to provide information necessary for breeding for resistance to septoria nodorum blotch, caused by Leptosphaeria nodorum, and to tan spot, caused by Pyrenophora tritici-repentis, in wheat.

Cultivars from different countries were screened for resistance to these pathogens in seedling tests conducted under controlled environmental conditions. The proportion of resistant cultivars identified was low; 12% of 482 tested were resistant to L. nodorum and only 2% of 350 cultivars tested were resistant to P. tritici-repentis. Resistance to L. nodorum was found also in alien species Triticum dicoccum, T. monococcum, T. tauschii and T. timopheevii. The resistance of accession PI 289599 of T. timopheevii has been transferred to a durum wheat background. Cultivars 7990-244A, 81IWMMN 2095, Erik, Hadden and ZG 7581-83 are resistant to both pathogens. Most resistant cultivars were winter wheats and the level of resistance in spring wheats was low. An important exception is spring wheat cultivar 86ISMN 2137 which is highly resistant to L. nodorum. All currently registered western Canadian wheat varieties were susceptible to both pathogens.

The inheritance and heritability of resistance was studied in controlled environment seedling tests using crosses between resistant and susceptible parents. Parents resistant to L. nodorum were 71-10-12, 81IWMMN 2095, 82IWMMN 2019, 83IWMMN 2051, Coker 76-35, EA 13, EE 8, Hadden, Missouri Queen, Moking, Red Chief and ZG 7581-83. Resistance was controlled by a single gene in all crosses and was recessive or partially recessive in most crosses. Allelism tests showed that 81IWMMN 2095 and 82IWMMN 2019 likely possess the same gene for resistance and that this differs from the gene in Coker 76-35. Broad-sense heritability estimates ranged from 71% to 90%.

Cultivars 81IWMMN 2095, Hadden and ZG 7581-83 were found to carry single genes for resistance to P. tritici-repentis. In the crosses studied, resistance was recessive and broad-sense heritability estimates ranged from 79% to 95%.

Physiologic specialization in L. nodorum was studied in controlled environment tests by inoculating a set of cultivars with a range of isolates collected from different areas in Saskatchewan. A significant isolate x cultivar interaction was detected but appeared due mainly to the aberrant behaviour of one cultivar. While differences in the pathogenicity of isolates were measured, no clear evidence for the existence of physiologic races of this pathogen were found. Cultivar BIIMMMN 2095 was highly resistant to all isolates used whereas other cultivars varied in their level of resistance. Isolates from the North Battleford and Shellbrook areas were the most pathogenic. Insufficient data are available to determine whether there is an association between isolate pathogenicity and area of origin. However, this study indicates the desirability of testing resistant cultivars with a range of isolates to determine their value as sources of resistance.

Field tests were conducted in 1988 and 1989 at Saskatoon and Weirdale to study the association of resistance with agronomic traits and to relate seedling and adult plant disease reaction. Despite artificial inoculation and irrigation, disease epidemics could not be established at Saskatoon. However, a low level of disease developed from natural infection at Weirdale and disease rating was possible. A good relationship was found between field and seedling ratings, thus confirming the validity of using seedling testing to study and breed for resistance. A general association between resistance and tallness and late maturity, but not with growth habit, was found.

These diseases are major diseases of wheat in Saskatchewan and have increased in occurrence in recent years due largely to more continuous cropping and reduced tillage operations. In north-central regions yield losses attributed to these diseases are 14 - 15%, even at moderate disease levels. Development and use of resistant varieties will minimize these losses, thus enhancing the competitiveness of Saskatchewan wheat producers, and provide an environmentally safe method of control.

The results of this project provide the basis for such development. The resistance sources, simple genetic control, high heritability and good

correspondence between seedling test and field reaction are factors which will facilitate breeding for resistance which this project has already commenced. Such breeding efforts are underway with the resistance gene from [illegible] 2095 being transferred into Kenyon and lines should be available for field evaluation in 1991. Results to date indicate that resistance can be maintained and easily identified in a backcrossing program.

This study also is the first to clearly demonstrate monogenic control of resistance to L. nodorum and thus represents an important scientific contribution in this area.

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